Eczema / Dermatitis

Definition: non-specific inflammatory response of the skin to a variety of agents which may act on the skin → from outside (or) → from inside

Types:
- Exogenous
- Endogenous

→ Acute
→ Subacute
→ Chronic

Clinical features:
- **symptoms:** itching
- **signs:** ill-defined polymorphic eruption
  - 1st → erythematous macules / papules / vesicles
  - 2nd → oozing / crustation / scaling
  - 3rd → lichenification / fissuring

Histopathology:

① **Acute** → spongiosis (inter-cellular oedema)
ballooning (intra-cellular oedema)
→ intra-epidermal vesicle
upper dermis → mononuclear cells → may migrate into Epidermis → (Exocytosis)
* spongiotic dermatitis

② **Subacute** → mild acanthosis, parakeratosis, crust
* psoriasiform-spongiotic dermatitis

③ **chronic** → marked acanthosis, elongation of rete ridges
hyperkeratosis, parakeratosis
* psoriasiform dermatitis
+ Perivascular inflamatory infiltrate of histiocyte & esinophil in upper dermis & fibrosis (dermal fibrosis)

Spongiosis: Lymphocytic

MF (epidermotropism)

ROX

eczema

Eczema

Endogenous

1. Atopic dermatitis
2. pemphigyx
3. pityriasis alba
4. Seborrheic Ø
5. Statis (gravitational) Ø
6. Discoid (Nummular) eczema
7. Exfoliative dermatitis (Erythroderma)
8. hand eczema + eyelid eczema

Exogenous

1. Contact Ø
   - → 1ry irritant
   - → 2ry allergic
2. Infectious (eczematoid) Ø
3. Napkin dermatitis
4. eczematous polymorphic light eruption

* Atopic Dermatitis AD

* Definition:

Atopy → genetically-determined disorder characterized by excess IgE production

Atopic disorders → AD
- Bronchial asthma
- Allergic rhino-conjunctivitis (hay fever)

AD → endogenous eczema characterized by recurrent pruritic symmetric eruption

* classifications:

- Early-onset (before 2 years) & Late-onset (after puber
- Exogenous (Extrinsic) & Endogenous (Intrinsic)

↓ high IgE — 70-80 %

↓ normal IgE — 20-30 %

Date: / / no: 5

Clinical picture — 3 stages

1. **Infantile stage:** (age: 2 months – 2 years)
   - lesion: ①, ②
   - ① * edematous erythematous patch
   - site * affects mainly cheeks (F2°)
   - ② * covered with vesicles → exudative / crust
2. **Childhood stage:** (age: 2 years – 12 years)
   - * affects mainly flexural sites (F2°) (neck, popliteal & cubital fossa)
   - lesion * papules & lichenified lesions
3. **Adulthood stage:** (adults)
   - as childhood phase
   - may affect face & trunk

N.B

* Symptoms → pruritus
* special types → [redacted]
* Childhood phase = Besnier's prurigo (F2°)

Histopathology

According to stages يوصف بالتفصيل

Diagnostic criteria

Major criteria MUST have ③ out of ④

سؤال مهم جدًا

① pruritus (itch)

② chronic or chronically-relapsing dermatitis

③ personal or family history of (other) atopic diseases
e.g. Bronchial asthma / allergic rhino-conjunctivitis

④ Typical morphology & distribution →
facial (infant) — flexor (child) — face (adult)

Date: No: 6

[redacted]

1- Xerosis
2- orbital darkening
3- Dennie-Morgan infra-orbital fold
4- Keratoconus
5- Anterior subcapsular Cataract
6- Recurrent conjunctivitis
7- Cheilitis
8- Food intolerance
9- pityriasis alba
10- Facial pallor / erythema
11- [redacted]
12- [redacted]
13- [redacted]
14- [redacted]
15- Tendency to skin infections eg: Staph aureus & herpes simplex "Eczema herpeticum"
16- peri follicular accentuation (keratosis pilaris)
17- white dermographism / delayed blanch
18- Ichthyosis / palmer hyperlinearity
19- Early age of onset
20- Elevated IgE level
21- +ve Immediate type I skin test
22- Course influenced by emotional / environmental
23- Non-specific hand / foot dermatitis

Must have (3) or more

Date: no: 7

Pathogenesis of AD

① Genetic factors:

evidence: Concordance rate in monozygotic twins > dizygotic twins
* family history of AD & other atopies

② groups of genes:

* Genes encoding Epidermal proteins
  - mutations in fillagrin gene (FLG)
  - protein that aggregates Keratin filaments during terminal differentiation
  - Epidermal barrier dysfunction
  - ↑ transepidermal water loss
  - dry scaly skin (xerosis)

* Genes encoding immunologic proteins
  e.g.
  - cytokines
  - Toll-like receptors (TLRs)
  - immunological dysfunction

② Immunological dysfunction:

- Acute AD → Th2-mediated (IL4, 5, 13) → ↑IgE, ↓INFγ, ↑CMI
- Chronic AD → Th1-mediated (IL 2, TNFα, INFγ)

③ Environmental factors:

④ Psychological factors

I. Role of S. aureus: ↑ Colonization due to loss of epidermal barrier
↓ Antimicrobial peptides (hBD2 defensins)

II. * ↓↓ sweating → aggravate

Staph. aureus contribute sensitization & inflammation through:
1- Recognition of cell wall by TLR 2 → ⊕ inflamatory proces
2- Super Ag amplify immune responce
3- staph aureus toxin

S. aureus contribute to sensitization & inflamation through:
1. recognition of cell wall components by TLR-2 → stimulate inflammatory process
2. Superantigens amplify immune responses
3. toxins of S. aureus

① Infections: bacterial → S. aureus
→ Streptococcus pyogenes } impetiginization
viral → H.S.V → Eczema herpeticum
→ Molluscum contagiosum → widespread infection
② Eye complications: (minor criteria Hanifin)

Treatment

① Avoidance of triggering factors:

- Allergens (house dust mites, pollen, animal fur ....)
- Sweating
- Soaps & detergents
- wool / synthetic / rough fabrics
- Cigarette smokes
- Emotional stress
- foods e.g. fish banana nuts ....

② Symptomatic / Supportive T.R: (Skin care)

* Emollients (topical) * oil in bath
* non-alkali soaps * Topical antibiotics / antivirals
* Sedating antihistamines (for pruritus)

② Topical Treatment:

- Emollients ((BASIC TREATMENT))
- Steroids
- Calcineurin inhibitors e.g. pimecrolimus (Elidel)
- antibiotics
- antivirals

③ Systemic therapy:

- Systemic steroids → short term use only in severe flares
- Ciclosporin
- MTX
- Azathioprine
- Mycophenolate Mofetil
- IV Ig
- photo (chemo) therapy
- Biological therapy

R4:-

① Education:-

education of Parents that The disease is chronic & recurrent & required special care.

② General measures:-

avoidance of triggers →
- house dust, pollen, animal fur — "Inhalent allergen"
- Fish chocolate, strawberry ---- "Ingestant allergen"
- wool, Animal fur, hair — "Contactant allergen"
- Soap & detergent → use instead Non alkali Soap
- cigarette Smoking
- Emotional stress
- Rough fibres

③ R4 of attack:-

Topical →
- Emollient "Basic R4"
- steroid
- Calcineurin inhibitors
- Antibiotic
- Anti viral
- Antifungal

systemic → In No Response to topical →
- Syst. steroid → short term use only in severe
- Ciclosporin
- MTX
- Azathioprin
- Micophenolate Mofitil
- IVIg
- Photo therapy
- Biological therapy

Sedating Antihistaminic

④ R4 of Complications:-

Infection

Prophylactic Na hypochlorite

R4 if infection by AB & Antiviral "IV"

Date: ___ P.O: 10

Contact Dermatitis (CD)

- **Definition:** non-specific inflammatory reaction of the skin produced by substances coming in contact with the skin (i.e. exogenous eczema)
- **Types:** ① Irritant CD
  ② Allergic CD

Primary irritant CD	Secondary allergic CD
Causative agent: irritant substance	allergen (sensitizer)
e.g. * Alkali → soap, detergents, bleachings	e.g. Nickel → jewellery, watches, scissors, belts, cell phones
* Acids → HCl, H_2SO_4	
* Insecticides	
* Metal salts	potassium dichromate → tanners, painters, brick-cement
Mx: the irritant MUST be applied to the skin for [redacted]	topical antibiotics → neomycin, bacitracin
	Hair dyes, Shampoos, soaps, fragrance ...
	latex rubber
clinical features	
* occurs in all individuals	* only in individuals sensitive to allergen
* occurs since first exposure → rapid-onset	* occurs upon second/subsequent exposure → delayed-onset
* [redacted] to site of application	* May be [redacted] away from site of application

ICD	ACD
Pathogenesis: irritant → direct damage of KCs → e.g. eicosanoids from cell walls → attract neutrophils / lymphocytes → inflammatory response	allergen → uptaken by LC → regional LNs → activation of naive T-cell → sensitized T-cells → skin-homing → site of allergen
No antigen-presentation	Subsequent exposure → Type III hypersensitivity → Massive inflammatory reaction → Spongiosis, cell injury & cell death. i.e. Eczema
Langerhans cells: ↓↓	Langerhans cell: ↑↑ no. & function
chronic irritant dermatitis: Repeated exposure to weak irritant substance over long period e.g. House wife dermatitis	

Clinical picture of CD: acute, subacute, chronic

Histopathology of CD:

TR of CD:

1. Identify / exclude / Avoid triggering agents
2. Antihistamines to ↓↓ itching
3. Topical steroids: gel → acute, cream → subacute, oint → chronic
4. Topical emollients / soothing agents
5. Antibiotics if ↑↑ bacterial infection

(I) Immunosuppressive effect
(II) anti-inflammatory
(III) Antiproliferative i.e. anti mitotic ↓ DNA synthesis

In case of ICD:

Irritant substance → Keratinocyte → eicosanoid → phospholipase A2 → Arachedonic → Cox / Lox → leukotrienes → bronchospasm

② ⊖ phospholipase A2 → ⊖ Arachedonic → cyclooxygenase Cox → ↓ PG → ↓ Inflammation & Pain; Lox → lipoxins → ↓ leukotrienes → ↓ bronchospasm

③ Vaso Constriction of capillary bed

Scanned by CamScanner

Patch Test :

- The aim : to produce original eruption in miniature.
- Procedure :

  - The tested substance is applied "preferably diluted" to the back of the patient for 24-48h.
  - In finn chamber test, a shallow aluminium cup holding the test substance is fixed to the skin with tape.

- Results :

  - Reading of the patch started 1H, 1,2,3 days after removal of the patch.
  - Latent reaction may be observed 6 days or even 3weeks after removal of the patch indicate → that the patient develop hypersensitivity during applications of the test.
  - +ve allergic reactions :

    + (weakly +ve) → discrete papules + erythema.

    ++ (strongly +ve) → infiltrated papules + vesicles + Erythema.

    +++ (extracanly +ve) → coalescing vesicles & Bullae.
  - Allergic reaction extended beyond site of application of the tape.
  - Irritant reaction → sharply out line fade within 24 h. Limited to site of contact
  - Allergic and irritant presence may occur simultaneously.

Wood's light -ve

fluorescence

Seborrheic dermatitis

"subacute"

Def chronic inflamatory disease of the skin characterized by :-
1- Sharply marginated lesion covered é Greasy Yellowish or white scales
2- Located to areas of maximum sebacious activity e.g scalp, eyebrow, Nasolibial fold, Inter scapular area, chest, groin, axilla, ears

Cause unknown but may be d.t :-
- Androgen dependant
① Endocrinal dysfunction
② organism → Malassezia furfur "Pit. ovale"

Clinical picture

① Pityriasis capitis :- The mildest & Commonest form.
occuring as → Dandruff ↔ Perifollicular redness & scaling
↔ may be sever extend to ↔ frontal hair line, Corona seborrhoica, post auricular
↓ complain → Itchy scalp
→ oily type → Pityriasis oleosa

② More sever cases :-
diffuse erythema & greasy scales affect other seborrheic area → eye brow, Nasolibial fold, eyelashs, beard, ears, orifis, flexural areas

③ Generalized form :-
Exfoliative Erythroderma

Histopathology subacute Eczemes

DD → Causes of scaly scalp → PRP, Psoriasis, T. Capitis
→ Causes of Erythroderma → MF & etc